surely warranted, as stated by Mehta and Pae.[1] Theoretically, inflow valve dysfunction may occur as well, even if it seems unlikely. The metallic sheath has since been redesigned and is now less mobile, because it is fixed to another metallic ring by a string.

In conclusion, rupture of the inflow conduit close to the inflow valve is a life-threatening complication of the TCI system with the mobile cage support, which occurs more often than expected. Possible mechanisms include multiple bending limited by the metallic cage support resulting from suboptimal placement, while the valvular anulus might be acting as an anchor. Patients with possible bleeding should receive aggressive treatment, even though exchange and replacement of the inflow conduit is a demanding task. The metallic sheath has recently been redesigned, and future patients may no longer be at risk for this complication.

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NODULAR PULMONARY AMYLOIDOSIS

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Pulmonary amyloid deposition generally occurs with concurrent primary systemic amyloidosis. Localized forms of pulmonary amyloidosis are rare and appear most frequently as an incidental lower lobe finding in chest radiographs. Tissue for diagnosis can be acquired by bronchoscopic, computed tomography–guided, thoracoscopic, or open biopsy techniques. Our patient had a nonproductive cough and multiple new lung nodules that led to open biopsy and diagnosis.

Although pulmonary amyloid is frequently found at autopsy as a sequela of primary systemic amyloidosis, cardiac failure resulting from amyloid deposition contributes to the patient's death in the majority of cases. Median survival time for 21 patients with this condition in a series reported by Utz, Swensen, and Gertz[1] was 16 months. Twelve percent to 25% of patients with systemic amyloid disease have multiple myeloma.

Pulmonary amyloid deposition not associated with systemic disease occurs in three anatomic distributions: tracheobronchial, parenchymal, and senile (or diffuse). Dyspnea, cough, and hemoptysis are symptoms of tracheobronchial amyloidosis, with the average patient age being between 53 and 62 years. Management strategies include observation, bronchoscopic resection, laser ablation, and lung resection. The senile or diffuse form is generally asymptomatic. In one autopsy series[2] diffuse amyloidosis was present in approximately 10% of patients older than age 80 years and in 50% of patients older than age 90. The localized parenchymal form of the disorder is found in fewer than half of all patients with isolated pulmonary amyloid.

Clinical summary. A 65-year-old woman had had a nonproductive cough for 2 months. She denied fever, night sweats, weight loss, and hemoptysis. Past medical history included mild hypothyroidism treated with oral levothyroxine, no exposure to chemicals or asbestos, and a history of working as a registered nurse in a tuberculosis sanitorium in the 1950s, with a positive purified protein derivative (PPD) tuberculin skin reaction when last tested in 1968. A chest radiograph performed 4 years before presentation in preparation for an orthopedic procedure showed several small

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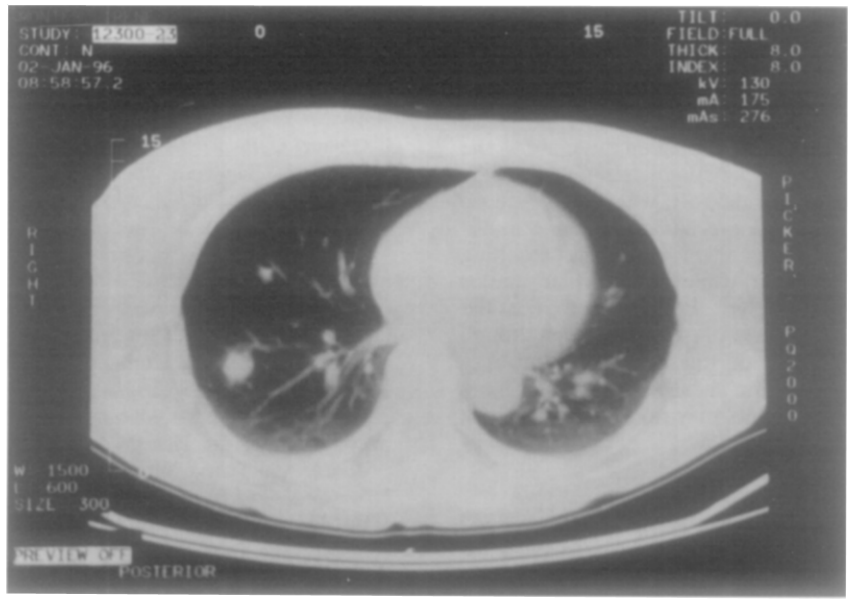

Fig. 1. Computed tomogram of the chest with intravenous contrast material showing eccentric soft tissue growth around the right lower lobe lesion.

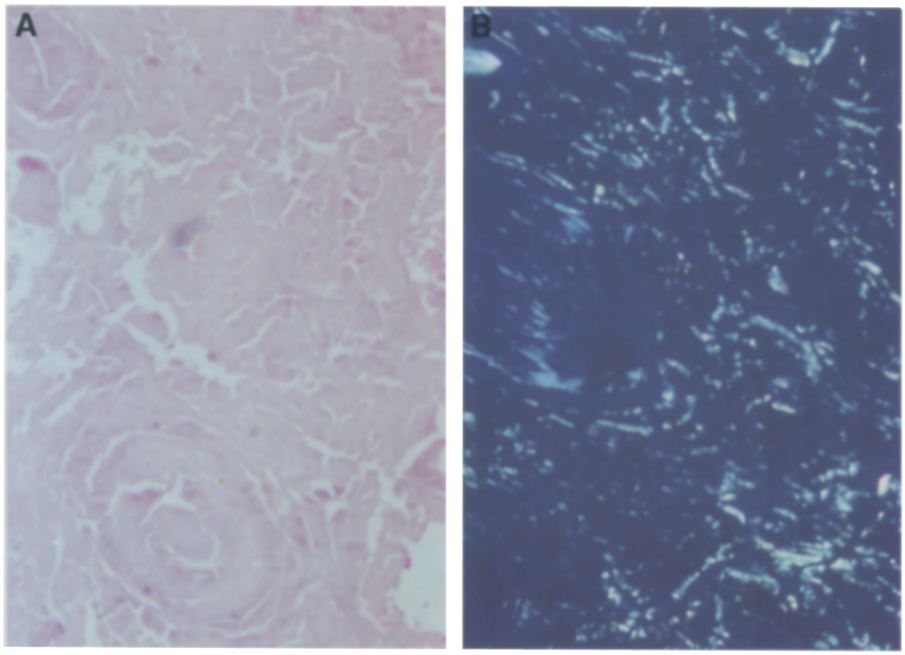

Fig. 2. A, Photomicrograph of right lower lobe mass demonstrating replacement of lung tissue with acidophilic homogeneous material and scattered lymphocytes. (Hematoxylin-eosin stain; original magnification ×300.) **B,** Amyloid deposits show apple-green birefringence and light polarization. (Congo red stain; original magnification ×500.)

calcified lung nodules with bilateral apical scarring but no mediastinal enlargement.

The current chest radiograph showed a 2 cm calcified nodule in the right lower lobe with smaller nodules in the right and left upper lobes. Computed tomograms of the chest demonstrated the multiple aforementioned calcified nodules and a dominant mass in the right lower lobe with eccentric soft tissue growth, but no

evidence of mediastinal adenopathy (Fig. 1). Mammography demonstrated benign coarse calcifications without evidence of neoplasia. Pulmonary function tests yielded a forced expiratory volume at 1 second of 2.75 L, and arterial blood gas measurements with the patient breathing room air showed a pH of 7.44, an oxygen tension of 101 mm Hg, and a carbon dioxide tension of 33 mm Hg. The PPD skin test remained positive and acid-fast bacillus sputum cultures were negative. Bronchoscopic studies showed no abnormalities, and brushings sent for cytologic examination were negative for malignancy.

After placement of a thoracic epidural infusion catheter and induction of general anesthesia with placement of a double-lumen endotracheal tube, a standard right posterolateral thoracotomy was performed through the fifth intercostal space. Fine adhesions between the upper lobe and apex were divided with cautery. Wedge resection of two small upper lobe nodules showed hyalinized, calcified granulomas on frozen section. The larger nodule, which was grayish red, was a poorly circumscribed mass formed by acidophilic staining acellular material on routine section (Fig. 2, *A*). Congo red staining of this material demonstrated apple-green birefringence and light polarization (Fig. 2, *B*), confirming the pathologic diagnosis of nodular pulmonary amyloid deposition.

Comment. The appearance of a new lung nodule with focal eccentric soft tissue growth in a patient with a history of tuberculosis exposure raises the possibility of scar carcinoma arising from a previous granuloma. Computed tomography-guided biopsy might have confirmed the diagnosis. Its negative predictive value is low, however, and multiple nodules necessitating evaluation were present throughout the lung. Open thoracotomy provided optimal access for this deep parenchymal lesion and ready availability for biopsy of other nodules. Furthermore, if a malignant tumor had been present, lobectomy or possibly pneumonectomy with lymph node dissection would have mandated open thoracotomy.

Exclusion of metastatic disease in this patient's work-up included a thorough breast examination and mammogram. Results of liver function tests were within normal limits, and urinalysis showed no hematuria or proteinuria. After diagnosis of nodular amyloid deposition, multiple myeloma screening was performed; urinary assay for Bence-Jones proteins was negative. The finding of normal blood gases and pulmonary function suggested against interstitial lung disease.

Treatment of nodular pulmonary amyloidosis as an isolated disease entity has been described in surgical reports,[3, 4] and such nodules have occurred in patients with cardiac amyloid[5] and after heart transplantation for cardiac amyloid.[6] Although solitary pulmonary parenchymal amyloid deposits have an overall benign course, those lesions located in the tracheobronchial tree often require laser resection and occasionally lobectomy. Large symptomatic amyloid deposits compressing adjacent mediastinal structures might also require excision.

Should isolated pulmonary amyloid deposits in the absence of systemic disease be treated with surgical resection? Even if a lesion is amenable to transbronchial biopsy, the diagnosis of pulmonary amyloid does not preclude existence of coexisting local or systemic malignant disease. The presence of amyloid serves only as a nonspecific marker of abnormal tissue metabolism and should alert the surgeon to the need for further diagnostic evaluation and open resection.

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